integrity Research Journals

Journal of Animal Science and Veterinary Medicine
Volume 10(3), pages 270-277, June 2025
Article Number: 4E2252896
ISSN: 2536-7099
https://doi.org/10.31248/JASVM2025.558
https://integrityresjournals.org/journal/JASVM

Review Article

Rotavirus in pigs: Review on structure, epidemiology, risk factors and zoonotic potential

Evalyne Chepkirui Bett[1]* and Wyckliff Ngetich[2]

[1]Department of Veterinary Public Health, Pharmacology and Toxicology, University of Nairobi. P.O Box 29053-00625, Kangemi, Kenya.
[2]Department of Veterinary Surgery, Theriogenology and Medicine, Egerton University. P.O Box 536-20115, Egerton, Nakuru, Kenya.

*Corresponding author. Email: echepkirui@gmail.com; Tel: +254708627748.

Received 4th April 2025; Accepted 16th June 2025

ABSTRACT: Pig production is an important livestock subsector contributing immensely to food security and household income generation, especially in developing countries. Rotavirus infections, which are the leading cause of dehydrating diarrhoea in piglets, hinder their productivity. To date, there are ten Rotavirus groups (A-J) detected and distributed in both humans and animals, with some strains demonstrating zoonotic potential. Some of the risk factors of Rotavirus infection in pig farms include: age of pigs, herd size, presence of other animals, farm management practices and overall sanitation. Evolution of these viruses, especially through reassortment events, has led to the development of novel genotypes with implications for both animal and human health. Rotaviruses are genetically diverse and persist in the environment, and therefore, frequent surveillance, especially molecular characterisation, is of vital importance in the mitigation of these viruses. This review highlights the structure of Rotaviruses, epidemiologic surveillance efforts, identified risk factors, and the potential for interspecies transmission. It also aims to identify research gaps, particularly regarding antigenic variation of circulating strains in pigs within developing countries. Improved surveillance and control measures are essential for enhancing pig health and maximising the economic benefits of pig production.

Keywords: Epidemiology, structure, risk factors, Rotavirus.

INTRODUCTION

Pig farming plays a critical role in global food systems, with pork representing one of the most accessible and affordable sources of protein for millions of people. As global demand for pork continues to rise (FAO, 2021), the swine industry faces significant challenges, particularly from infectious diseases such as Rotavirus, which are major contributors to production losses. In neonatal and weaned piglets, Rotavirus infections are a leading cause of diarrhoea, characterised by high morbidity rates. This condition hampers growth performance and leads to substantial economic losses across the industry (Burrough, 2021; Saif and Vlasova, 2022).

Rotaviruses, members of the *Sedoreoviridae* family, are environmentally resilient, capable of withstanding a wide range of temperatures, low pH levels, and common disinfectants. This resilience contributes to their widespread distribution in pig-rearing environments (Matthijnssens *et al*., 2022; Saif and Vlasova, 2022). To date, five Rotavirus groups A, B, C, E, and H have been identified in pigs (Vlasova *et al*., 2017). While most research has focused on Group A, interest in the remaining groups is gradually increasing. Despite sharing antigenic properties, these groups do not provide cross-protective immunity, meaning that protection against one group does not confer immunity to others (Qiao *et al*., 2024; Park *et al*., 2019).

Understanding the genetic diversity among porcine Rotaviruses is therefore crucial for the development of effective and targeted vaccines. This review explores the current knowledge on the different Rotavirus groups

affecting pigs, their epidemiological characteristics, and their implications for vaccine design. Recent findings, including those by Qiao *et al*. (2024), have identified porcine rotavirus strains with high genetic similarity to human strains, suggesting potential interspecies transmission through reassortment events (Amimo *et al*., 2015; Wu *et al*., 2017). These findings underscore the importance of ongoing molecular surveillance to inform effective prevention and control strategies in both veterinary and public health contexts.

ROTAVIRUS IN PIGS

Structure of rotavirus

Rotaviruses are members of the *Sedoreoviridae* family and exhibit a wheel-shaped appearance when viewed under electron microscopy (Matthijnssens *et al*., 2022; Ferrari *et al*., 2022). This characteristic appearance explains the origin of the name "Rotavirus," as *rota* means "wheel" in Latin (Chang *et al*., 2012). These viruses are non-enveloped, double-stranded RNA viruses with a three-layered capsid and a diameter of approximately 70 nm. The capsid encloses 11 genome segments of double-stranded RNA (Vlasova *et al*., 2017).

These segments encode six non-structural proteins (NSP1–NSP6) and six structural viral proteins (VP1, VP2, VP3, VP4, VP6, and VP7). Viral proteins VP1 to VP3 form the inner capsid, VP6 constitutes the middle capsid, and VP4, along with VP7, are located on the outer capsid (Crawford *et al.*, 2017; Matthijnssens *et al*., 2022). VP1, VP2, and VP3 are involved in transcription and replication, while VP6 determines the Rotavirus group. VP4 and VP7 play crucial roles in viral attachment to host cells and in inducing a humoral immune response (Crawford *et al*., 2017). Additionally, VP4 and VP7 are used to determine the P (protease-sensitive) and G (glycoprotein) genotypes of Rotavirus, respectively (Papp *et al*., 2013).

Non-structural protein 1 (NSP1) is involved in evading the host immune response, allowing the virus to enter intestinal cells and replicate (Amimo *et al*., 2021). Non-structural protein 4 (NSP4) acts as an enterotoxin and contributes to the pathogenesis of the infection (Estes and Greenberg, 2013).

It has been reported that nearly every pig is exposed to Rotavirus infection at some point in its lifetime, with older pigs often experiencing mixed-strain infections (Homwong *et al*., 2016). Naïve adult pigs and suckling piglets are more susceptible to Rotavirus infection due to their immature or unprimed immune systems. However, adult pigs generally develop resistance to infection through immunity acquired from natural exposure (Amimo *et al*., 2013). Piglets and young pigs that receive inadequate colostrum are particularly vulnerable to Rotavirus infection because they lack sufficient passive immunity (Shepherd *et al*., 2019). Similarly, piglets in early life lack adequate immunoglobulins, making them more susceptible to viral infections, as they are unable to mount a rapid and robust immune response (Chepngeno *et al.,* 2019).

Classification of rotavirus

To date, there are 10 Rotavirus groups (A-J) that have been detected in both humans and animals, with groups A, B and C as the most common (Amimo *et al.,* 2013). This classification is based on the gene sequencing of viral protein 6 (VP6) as well as full genomic sequencing (Ferrari *et al*., 2022). Among these groups, Rotavirus A (RVA) has been reported to have the highest prevalence in both humans and animals worldwide (Bányai *et al.,* 2017).

Rotaviruses have been reported globally as the major causes of viral gastroenteritis in pig farms. Five geno-groups (RVA, RVB, RVC, RVE and RVH) have been detected in pigs (Vlasova *et al*., 2017). They cause significant economic losses to pig farmers due to high morbidity and mortality of piglets, retarded growth of the infected pigs and high cost of treatment (Amimo *et al*., 2015).

Rotavirus A (RVA) has been reported to be the most frequent strain associated with porcine diarrhoea (Vlasova *et al*., 2017; Marthaler *et al*., 2014a), causing about 90% of Rotavirus infections (Wu *et al*., 2022). Its prevalence is high in nursing and postweaning pigs (Ferarri *et al*., 2022). A study by Ferarri *et al*. (2022) reported a prevalence of 53% in Northern Italy, while Amimo *et al.* (2015) reported 26.2% along the Kenya and Uganda border. The number of samples in each study could be the reason behind the difference in prevalence. The G and P genotypes that are common for RVA include G3-5, G9, G11, P6, P7, P13 and P19 (Amimo *et al*., 2015). Rotavirus A strains are diverse, and their infections are widespread and endemic in both clinical and asymptomatic piglets (Amimo *et al*., 2015). For instance, in the United States, the most predominant genotype in 2012 was G9P (Amimo *et al*., 2013), but in 2021, the predominant genotype was G5P (Doerksen *et al*., 2022).

Rotavirus B (RVB) is the most diverse group of Rotaviruses and has 20G genotypes (Shepherd *et al*., 2017; Vlasova *et al*., 2017). It co-infects piglets with RVA and RVC, suggesting that RVB could be a secondary pathogen (Marthaler *et al*., 2014a). A study by Miyabe *et al*. (2020) reported RVB as the primary pathogen with a prevalence of 71.1% in newborn piglets. However, Ferarri *et al*. (2022) reported a higher prevalence in fattening pigs (46.42%) compared to suckling (20.75%) and weaning piglets (43.93%), suggesting exposure increases with age of the pig.

Rotavirus C (RVC) has been reported in different countries worldwide (Tuanthap *et al.*, 2018). This group has been detected from all ages of pigs (Marthaler *et al.,* 2013). Nine G genotypes and 7 P genotypes have been molecularly characterized (Tuanthap *et al.,* 2018). From

the sequence analysis of the VP6 gene, 11 I genotype have been identified (Kattoor *et al*., 2017). Chepngeno *et al*. (2019) reported a prevalence of 79.1% of RVC RNA in nursing piglets, with more infections in clinical than in asymptomatic piglets.

From the sequence of VP4 and VP7 genes, Suzuki and Inoue (2018) identified 6P and 10G genotypes, respectively, in Japan and from the NSP1 gene, they revealed the presence of 6 genotypes (A1-A6). The first complete genome of RVH was done in South Africa, and it showed a close relationship with the Brazilian and Japanese strains (Nyaga *et al*., 2015).

Transmission

Rotavirus is very stable and persistent in the environment and can cause infection even under harsh conditions. Rotaviruses are very diverse due to genetic reassortment events that occur frequently (Vlasova *et al*., 2017). Transmission of these viruses is through the faecal-oral route (contact with contaminated water, feed and fomites), with several factors exacerbating the transmission process. Firstly, Rotavirus is shed in faeces, and one gram of faecal material contains a high concentration of the virus of about 10^{10} particles of infectious Rotavirus. Secondly, for infection to occur, less inoculum is required. Third, as the sows farrow, they shed the virus, increasing infection in piglets (Shepherd *et al*., 2019). The prolonged persistence of the virus in the environment increases the infection and the overall prevalence of the infection (Shepherd *et al*., 2019; Fongaro *et al*., 2015).

Pathogenesis

Viral infection starts with the attachment and entry of Rotaviruses into the epithelial cells (Cui *et al*., 2019). This virus enters the intestinal epithelial cells through either direct entry or epithelial endocytosis, as it is dependent on these cells for its transmission and replication (Amimo *et al.,* 2021). Rotavirus requires certain receptors in order to attach to the epithelial cells. These receptors include sialic acid, Histo blood group antigens (HBGAs), integrins, Heat shock cognate 70 protein (Hsc70), among other co-receptors (Cui *et al*., 2019; Amimo *et al.,* 2021). Many studies and experiments have been carried out, and others are still underway to try and understand the role of these receptors in Rotaviral infection. In a study carried out by Guo *et al.* (2021) using porcine crypt-derived 3D intestinal enteroids (PIEs), it was reported that different strains of RVA prefer certain types of HBGA and that sialic acid plays a role in attachment and replication of some strains of Rotavirus. Cell entry involves lysis of the outer proteins, attachment to the cell, followed by the digestion of the outer capsid and lastly entry of the Rotavirus double-layer particles into the cell cytoplasm. The study by Cui *et al*. (2019) used cultured porcine enterocytes to demonstrate the importance of intestinal epithelium integrity in defence against Rotavirus infection. The host's immune system counteracts it through different mechanisms such as mucus production, activation of signalling pathways like the toll-like receptor pathway and the RIG-I signalling pathway and chemical production such as cytokines (Amimo *et al.,* 2021). For a successful entry and replication into the epithelial cells, Rotaviruses have developed ways of evading those host immune mechanisms. For example, by use of NSP1, Rotaviruses can degrade the production of interferons, which are responsible for the innate immune response. The NSP1 also inhibits the production of proinflammatory chemicals responsible for apoptosis, causing Rotavirus to stay longer in infected cells (Arnold and Patton, 2011). Rotaviruses replicate in the mature enterocytes of the small intestines, causing atrophy of the microvilli (Chepngeno *et al.,* 2020). During infection, Rotavirus NSP4 disrupts the regulation of host cell calcium signalling pathways, leading to impaired homeostasis and hence secretory diarrhoea (Chang *et al*., 2020). Rotaviruses form clusters covered in vesicles, and they leave the host cells before lysis, therefore avoiding degradation (Santiana *et al*., 2018).

Several mechanisms contribute to diarrhoea development during the infection period. Destruction of enterocytes, ischemic villi and infected epithelial cells releasing vasoactive agents contribute to malabsorption, leading to diarrhoea (Vlasova *et al*., 2017). Another mechanism is that Rotaviruses have non-structural protein 4 (NSP4), which acts as a secretory agonist and an enterotoxin. This protein induces age and dose-dependent diarrhoea response by causing efflux of calcium from the endoplasmic reticulum. These calcium increases cell permeability and alteration of epithelial barrier integrity, causing secretory diarrhoea (Estes *et al*., 2001). The NSP4 protein also stimulates the enteric nervous system, increasing intestinal motility, contributing to diarrhoea (Estes *et al*, 2001). Opportunistic enteric pathogens that coexist with Rotavirus infection, such as *Clostridium*, *Escherichia coli*, *Salmonella*, Astrovirus, Coronavirus, and Noro-virus, enter due to the breakdown of intestinal mucosa and therefore complicate the infection (Chatzopoulos *et al*., 2013).

Rotavirus infections have been reported in both clinical and subclinical pigs (Amimo *et al*., 2015; Theuns *et al*., 2016). The clinical manifestations include: profuse watery diarrhoea, dehydration, vomiting and anorexia, weight loss, weakness and death. The severity of these signs depends on the age of the pig, immune status, farm herd health, Rotavirus strain and the presence of other bacteria (Shepherd *et al.,* 2019). When combined with enteric bacterial infection, the severity of Rotavirus infection increases (Theuns *et al*., 2014).

Diagnosis

Rotavirus infections exhibit clinical signs similar to those

caused by other enteric pathogens. Additionally, the detection of these infections in asymptomatic pigs necessitates laboratory testing for accurate diagnosis. Various methods are available to diagnose Rotavirus infections in animals, including electron microscopy, viral isolation, antigen detection assays such as enzyme-linked immunosorbent assay (ELISA), real-time reverse transcriptase PCR, and polyacrylamide gel electrophoresis (PAGE) (Shepherd *et al*., 2019). Faecal material is commonly used to detect either the double-stranded RNA of the virus or its antigens. Reverse transcriptase PCR is frequently employed as a diagnostic tool to quantify the concentration of the virus in faecal samples (Costantini *et al*., 2007). Moreover, multiplex RT-PCR has been developed using primers for several enteric viruses, enabling the differentiation of various Rotavirus strains (Theuns *et al*., 2016).

Management

There is no specific treatment for Rotavirus infections, and therefore, symptomatic and empirical treatment is required. Rehydration and feeding of the infected pigs with a high-energy diet is important to replace the lost fluids and nutrients. Antibiotics can be administered to control for co-infection with enteric bacteria. Some probiotics have been shown to reduce the severity of the infections and improve immune response by the intestinal mucosa (Vlasova *et al*., 2017).

The first physical barrier against the infection is the intestinal epithelial cells; therefore, the integrity of these cells is of paramount significance in host immunity (Holloway and Coulson, 2013; Amimo *et al*., 2021). Improving the host immunity is a way of controlling Rotavirus infections. A study by Mao *et al.* (2018) showed that dietary supplementation of feed with L-isoleucine improves piglets' immunity and growth. Isoleucine-fed piglets showed increased immunoglobulins and Rotavirus antibody levels, a sign of improved humoral immunity. The study also showed that isoleucine decreases NSP4 levels, therefore decreasing diarrhoea in piglets. Another study by Chepngeno *et al*. (2022) suggested that vitamin A supplementation and Rotavirus A inoculation of the sows during pregnancy and lactation elevate immune responses of the sow and passive immunity to the piglets. Tian *et al*. (2016) suggested that Vitamin D3 supplementation reduces Rotavirus infection in pigs through the cell degradation of infected cells. Vitamin D has been reported to activate the RIG-I signalling pathway, reducing the negative effects of Rotavirus infection, though the exact mechanism of action is not known (Lee, 2020**)**.

Maternal immunity is very important in the prevention of infection in piglets (Chepngeno *et al*., 2019). Therefore, piglets should receive enough quantities of colostrum to gain antibodies against the rotavirus infection. Sows' colostrum and milk contain Ig G and Ig A. Lack of colostrum and early weaning causes Rotavirus diarrhoea in piglets, showing the significance of maternal antibodies in early age. This passive immunity declines over time, requiring active immunity through vaccination (Nguyen *et al*., 2007).

Epidemiology

Rotaviruses, especially RVA and RVC, have been reported in most parts of the world (Vlasova *et al*., 2017). However, RVB has been reported in the United States, Japan, Russia, Switzerland and South Africa. Rotavirus H strain has been reported in Japan, Brazil, the United States of America and South Africa (Kumar *et al.*, 2022). Nyaga and colleagues did a complete genome analysis of the first RVH in South Africa in 2016. However, RVE has been reported only in the UK in the 1980s (Chasey *et al*., 1986), and no other literature has been published. Rotaviruses A and C have been extensively researched, unlike other genotypes (Amimo *et al*., 2021; Mao *et al*., 2018; Chepngeno *et al*., 2022). In African countries, there is scarce literature on porcine Rotaviruses due to a lack of surveillance systems. For instance, in Kenya, only one study has been published on porcine Rotaviruses since 2015, and that only detected RVA and RVC in backyard pigs. With the diversity and the zoonotic nature of Rotavirus, more research and surveillance should be put in place, especially for the other types (RVB, RVH and RVE).

Molecular tools have played a very important role in understanding the genetic diversity of Rotaviruses and their zoonotic potential. This is through the identification of different genotypes and their genetic relationships (Shepherd *et al*., 2019). Primers specific to the Viral Protein (VP4 and VP7) gene segments have been used to identify different G and P genotypes (Theuns *et al*., 2016). Twenty-seven different G genotypes and 37 different P genotypes have been identified for Rotavirus A in both animals and humans (Vlasova *et al*., 2017). Twelve G and 16 P genotypes have been identified in pigs (Amimo *et al*., 2015). A study by Monteagudo *et al*. (2022) in Spain revealed the following genotypes for RVA: G4, G9, G3, G5 and G11 for the VP7 gene and P7, P23, P6 and P13 for the VP4 gene. Amimo *et al*. (2015) detected P6, P8 and P13 genotypes from sequencing of the VP4 gene, where P6 and P8 had a close genetic relationship with the human strains. Whole genome classification of RVB in the United States of America identified 26G and 5P genotypes (Shepherd *et al.*, 2018). A recent study from China identified a new reassortant of RVB (HNLY-2022), which had caused an outbreak of piglet diarrhoea (Li *et al*., 2024). In a study in Thailand by Tuanthap *et al*. (2018), Rotavirus C, genetic analysis of VP7 and VP4 genes revealed the presence of nine G and seven P genotypes. Whole genome analysis of RVA strains in samples from sub–Saharan Africa has shown mixed infections with coinfect-

tion of porcine and bovine strains being reported (Nyaga *et al*., 2015). Rotavirus H (RVH) has been detected in pigs from Brazil, Japan, South Africa and the United States of America (Vlasova *et al*., 2017). In the United States of America, a prevalence of 15% was reported (Martheler *et al*., 2014b) and 9.4% in Brazil (Flores *et al*., 2021). It has been reported that RVH mostly co-infects with RVC (Suzuki and Inoue, 2018). In East Africa, Amimo *et al*. (2015) detected and characterised RVA strains in Kenya and Uganda with P6, P8 and P13 genotypes.

Risk factors

There are several factors that have been associated with the occurrence of Rotavirus infections in pig farms. These factors range from animal factors, farm-level factors, management factors, to seasonal factors. Animal factors include age, sex and breed. Piglets under four months have a higher risk of exposure to Rotavirus infection as compared to older ones (Amimo *et al*., 2017). Murao *et al*. (2019) reported a higher incidence of RVA in asymptomatic adult pigs, suggesting that adult pigs are asymptomatic carriers and hence they play a big role in the transmission of the virus to the piglets. Herd size, presence of other animals in the farm and husbandry systems are among the farm-level factors. Large herd size, presence of other animals such as goats, cattle and chicken, and free-range pigs have been reported to increase the risk of exposure to Rotavirus infections (Murao *et al.,* 2019). Management factors such as sanitation and waste disposal, source of feed, water and biosecurity measures in the farm influence the risk of Rotavirus infections in pig farms (Amimo *et al*., 2017; Murao *et al*., 2019). Similarly, weather conditions, especially cold months, have been associated with increased infections globally (Patel *et al.,* 2013).

A study in India showed that poor ventilation of pig pens, feeding of homemade feed and sourcing of water from shallow wells increases the risk of Rotavirus infections (VinodhKumar *et al*., 2020). Frequent change of ingredients in homemade feed leads to improper gut health. Shallow well water is easily contaminated by faecal matter, and hence, the increase in infections. A study from Nigeria implicated intensive pig farming systems, water sourcing from dams and mixed farming to higher rotavirus infections in pig farms (Delia *et al*., 2019).

Zoonotic potential

Rotaviruses have evolved through different mechanisms to form new genotypes, which may be of zoonotic importance. These mechanisms of evolution include point mutations, recombination and majorly reassortment (Collins *et al*., 2010). Rotaviruses are prone to reassortments due to the segmented nature of their genome, leading to the formation of new variants which may be more virulent than the parent gene (Malik *et al*., 2020). There has been development of uncommon/novel Rotavirus genotypes in the human population, and many of them have been reported to have originated from domestic animals (Cook *et al*., 2004).

Studies have shown that some of the Rotavirus A strains have genetic relatedness to human strains. Amimo *et al*. (2015) reported that P6 and P8 genotypes detected in pigs were genetically closely related to human strains and that there could be a possible interspecies transmission. Human Rotavirus A strain diversity shows susceptibility of humans to Rotavirus infections from animal origin (Doro *et al*., 2015). Rotavirus A G9 and G12 human genotypes have similarities with porcine G9 and G12, normally observed in piglets (Vlasova *et al*., 2017). Wu *et al.* (2017) reported RVA strains with high genetic similarity detected in children and pigs.

The suggested animal reservoirs for human Rotavirus infections include: porcine, bovine, rodents and ovine. Reports have described sporadic cases of human infections coming from different animal origins through interspecies transmission (Vlasova *et al*., 2017). Ten G genotypes and 7 P genotypes from porcine origin have been detected in humans (Doro *et al*., 2015).

The seasonal pattern for porcine RVA circulation resembles that of human RVA circulation, which occurs during the cooler months, suggesting that pigs could be the reservoirs for human infections (Patel *et al.,* 2013). Whole genome analysis of Rotavirus A from Moroccan nomadic livestock revealed that some livestock strains had similarities with the human ma31 strain, suggesting zoonotic transmission between livestock and humans (Alaoui *et al.,* 2020).

Rotavirus C has also been shown to be of zoonotic significance. There is evidence of genomic reassortments between human RVC and porcine RVC. Porcine RVC strains carrying human-like NSP4 and NSP5 have been detected (Costa *et al*., 2020). Another study by Kattoor *et al*. (2017) has shown a human-like VP6 gene in porcine RVC. There are shared HBGAs between humans and animals, suggesting a possible cross-transmission of Rotavirus strains between them (Jiang *et al.,* 2017).

CONCLUSION AND RECOMMENDATIONS

Porcine Rotavirus is widely distributed, although some regions have limited information or few studies on porcine Rotavirus. With the reassortment and the spillover to humans, more research and surveillance studies need to be carried out. In developing countries, animals and people live closely in most cases, share the same environment, making it easier for such pathogens as Rotavirus to move from animals to humans, yet fewer studies have been done on porcine Rotavirus. There is a need for more research and creation of awareness on

biosafety and biosecurity measures on pig farms. There is limited information, especially on the molecular characterisation of the circulating strains in developing countries. This is an informational gap that needs to be filled to understand the circulating strains in pig farms and take measures accordingly.

CONFLICT OF INTEREST

The author(s) declared no potential conflicts of interest concerning the research, authorship, finance, and/or publication of this article.

REFERENCES

Alaoui, A. S., Melloul, M., El Alaoui, M. A., Boulahyaoui, H., Loutfi, C., Touil, N., & El Fahime, E. (2020). Evidence for zoonotic transmission of species A rotavirus from goat and cattle in nomadic herds in Morocco, 2012–2014. *Virus Genes*, *56*, 582-593.

Amimo, J. O., Junga, J. O., Ogara, W. O., Vlasova, A. N., Njahira, M. N., Maina, S., Okoth, E.A., Bishop, R. P., Saif, L. J., & Djikeng, A. (2015). Detection and genetic characterisation of porcine group A rotaviruses in asymptomatic pigs in smallholder farms in East Africa: predominance of P [8] genotype resembling human strains. *Veterinary microbiology*, *175*(2-4), 195-210.

Amimo, J. O., Otieno, T. F., Okoth, E., Onono, J. O., & Bett, B. (2017). Risk factors for rotavirus infection in pigs in Busia and Teso subcounties, Western Kenya. *Tropical animal health and production*, *49*, 105-112.

Amimo, J. O., Raev, S. A., Chepngeno, J., Mainga, A. O., Guo, Y., Saif, L., & Vlasova, A. N. (2021). Rotavirus interactions with host intestinal epithelial cells. *Frontiers in immunology*, *12*, 793841.

Amimo, J. O., Vlasova, A. N., & Saif, L. J. (2013). Detection and genetic diversity of porcine group A rotaviruses in historic (2004) and recent (2011 and 2012) swine faecal samples in Ohio: predominance of the G9P [13] genotype in nursing piglets. *Journal of Clinical Microbiology*, *51*(4), 1142-1151.

Arnold, M. M., & Patton, J. T. (2011). Diversity of interferon antagonist activities mediated by NSP1 proteins of different rotavirus strains. *Journal of Virology*, *85*(5), 1970-1979.

Bányai, K., Kemenesi, G., Budinski, I., Földes, F., Zana, B., Marton, S., Varga-Kugler, R., Oldal, M., Kurucz, K., & Jakab, F. (2017). Candidate new rotavirus species in Schreiber's bats, Serbia. *Infection, Genetics and Evolution*, *48*, 19-26.

Burrough, E. (2021). *Rotaviral Enteritis in Pigs*. MSD Veterinary Manual.

Chang, K., Kim, Y., Saif, L. J. (2012) Rotavirus and reovirus. *Diseases of Swine*, *10*, 621-634.

Chang-Graham, A. L., Perry, J. L., Engevik, M. A., Engevik, K. A., Scribano, F. J., Gebert, J. T., Danhof, H. A., & Hyser, J. M. (2020). Rotavirus induces intercellular calcium waves through ADP signaling. *Science*, *370*(6519), eabc3621.

Chasey, D., Bridger, J. C., & McCrae, M. A. (1986). A new type of atypical rotavirus in pigs. *Archives of Virology*, *89*, 235-243.

Chatzopoulos, D. C., Athanasiou, L. V., Spyrou, V., Fthenakis, G. C., & Billinis, C. (2013). Rotavirus infections in domestic animals. *Journal of the Hellenic Veterinary Medical Society*, *64*(2), 145-160.

Chepngeno, J., Diaz, A., Paim, F. C., Saif, L. J., & Vlasova, A. N. (2019). Rotavirus C: Prevalence in suckling piglets and development of virus-like particles to assess the influence of maternal immunity on the disease development. *Veterinary Research*, *50*, 1-12.

Chepngeno, J., Takanashi, S., Diaz, A., Michael, H., Paim, F. C., Rahe, M. C., Hayez, J.R., Baker, C. & Vlasova, A. N. (2020). Comparative sequence analysis of historic and current porcine rotavirus c strains and their pathogenesis in 3-day-old and 3-week-old piglets. *Frontiers in Microbiology*, *11*, 780.

Collins, P. J., Martella, V., Sleator, R. D., Fanning, S., & O'shea, H. (2010). Detection and characterisation of group A rotavirus in asymptomatic piglets in southern Ireland. *Archives of Virology*, *155*, 1247-1259.

Cook, N., Bridger, J., Kendall, K., Gomara, M. I., El-Attar, L., & Gray, J. (2004). The zoonotic potential of rotavirus. *Journal of Infection*, *48*(4), 289-302.

Costa, F. B., Flores, P. S., Amorim, A. R., Mendes, G. D. S., & Santos, N. (2020). Porcine rotavirus C strains carrying human-like NSP4 and NSP5. *Zoonoses and Public Health*, *67*(8), 849-861.

Costantini, V. P., Azevedo, A. C., Li, X., Williams, M. C., Michel Jr, F. C., & Saif, L. J. (2007). Effects of different animal waste treatment technologies on detection and viability of porcine enteric viruses. *Applied and Environmental Microbiology*, *73*(16), 5284-5291.

Crawford, S. E., Ramani, S., Tate, J. E., Parashar, U. D., Svensson, L., Hagbom, M., Franco, M. A., & Estes, M. K. (2017). Rotavirus infection. *Nature Reviews Disease Primers*, *3*(1), 1-16.

Cui, T., Theuns, S., Xie, J., & Nauwynck, H. J. (2019). Porcine rotavirus mainly infects primary porcine enterocytes at the basolateral surface. *Veterinary Research*, *50*, 1-13.

Delia, T. A., Dzikwi-Emennaa, A. A., Kwaga, J. K. P., Kia, G. S. N., Olufemi, O. T., Ololorin, G. R., & Adanu, A. W. (2019). Prevalence of porcine rotavirus antigen and associated risk factors in pig-raising communities and institutional piggeries in Zaria, Kaduna state, Nigeria. *Folia Veterinaria*, *63*(1), 17-23.

Doerksen, T., Christensen, T., Lu, A., Noll, L., Bai, J., Henningson, J., & Palinski, R. (2022). Assessment of porcine Rotavirus-associated virome variations in pigs with enteric disease. *Veterinary Microbiology*, *270*, 109447.

Doro, R., Farkas, S. L., Martella, V., & Banyai, K. (2015). Zoonotic transmission of rotavirus: surveillance and control. *Expert review of anti-infective therapy*, *13*(11), 1337-1350.

Estes, M. K., & Greenberg, H. B. (2013). Rotaviruses. In: Fields, B. N., Knipe, D. M., & Howley, P. M. (eds.). *Fields virology* (pp. 1347-1401), 6th edition. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins.

Estes, M. K., Kang, G., Zeng, C. Q. Y., Crawford, S. E., & Ciarlet, M. (2001, May). Pathogenesis of rotavirus gastroenteritis. In *Gastroenteritis Viruses: Novartis Foundation Symposium, 238* (Vol. 238, pp. 82-100). Chichester, UK: John Wiley & Sons, Ltd.

Ferrari, E., Salogni, C., Martella, V., Alborali, G. L., Scaburri, A., & Boniotti, M. B. (2022). Assessing the epidemiology of rotavirus A, B, C and H in diarrheic pigs of different ages in northern Italy. *Pathogens*, *11*(4), 467.

Flores, P. S., Costa, F. B., Amorim, A. R., Mendes, G. S., Rojas, M., & Santos, N. (2021). Rotavirus A, C, and H in Brazilian pigs: Potential for zoonotic transmission of RVA. *Journal of Veterinary Diagnostic Investigation*, *33*(1), 129-135.

Fongaro, G., Padilha, J., Schissi, C. D., Nascimento, M. A., Bampi, G. B., Viancelli, A., & Barardi, C. R. M. (2015). Human and animal enteric virus in groundwater from deep wells, and recreational and network water. *Environmental Science and Pollution Research*, *22*, 20060-20066.

Food and Agriculture Organisation of the United Nations (FAO) (2021). Meat market review. Retrieved from http://www.fao.org/3/cb3700en/cb3700en.pdf.

Guo, Y., Candelero-Rueda, R. A., Saif, L. J., & Vlasova, A. N. (2021). Infection of porcine small intestinal enteroids with human and pig rotavirus A strains reveals contrasting roles for histo-blood group antigens and terminal sialic acids. *PLoS Pathogens*, *17*(1), e1009237.

Homwong, N., Diaz, A., Rossow, S., Ciarlet, M., & Marthaler, D. (2016). Three-level mixed-effects logistic regression analysis reveals complex epidemiology of swine rotaviruses in diagnostic samples from North America. *PLoS One*, *11*(5), e0154734.

Holloway, G., & Coulson, B. S. (2013). Innate cellular responses to rotavirus infection. *Journal of General Virology*, *94*(6), 1151-1160.

Jiang, X., Liu, Y., & Tan, M. (2017). Histo-blood group antigens as receptors for rotavirus, new understanding on rotavirus epidemiology and vaccine strategy: Rotavirus host receptor and vaccine strategy. *Emerging Microbes & Infections*, *6*(1), 1-8.

Kattoor, J. J., Saurabh, S., Malik, Y. S., Sircar, S., Dhama, K., Ghosh, S., Banyai, K., Kobayashi, N. & Singh, R. K. (2017). Unexpected detection of porcine rotavirus C strains carrying human origin VP6 gene. *Veterinary Quarterly*, *37*(1), 252-261.

Kumar, D., Shepherd, F. K., Springer, N. L., Mwangi, W., & Marthaler, D. G. (2022). Rotavirus infection in swine: genotypic diversity, immune responses, and role of gut microbiome in rotavirus immunity. *Pathogens*, *11*(10), 1078.

Lee, C. (2020). Controversial effects of vitamin D and related genes on viral infections, pathogenesis, and treatment outcomes. *Nutrients*, *12*(4), 962.

Li, Q., Wang, Z., Jiang, J., He, B., He, S., Tu, C., Guo, Y. & Gong, W. (2024). Outbreak of piglet diarrhoea associated with a new reassortant porcine rotavirus B. *Veterinary Microbiology*, *288*, 109947.

Malik, Y. S., Bhat, S., Dar, P. S., Sircar, S., Dhama, K., & Singh, R. K. (2020). Evolving rotaviruses, interspecies transmission and zoonoses. *The Open Virology Journal*, *14*, 1-6.

Mao, X., Gu, C., Ren, M., Chen, D., Yu, B., He, J., Yu, J., Zheng, P., Luo, J., Luo, Y., Wang, J., Tian, G. & Yang, Q. (2018). L-isoleucine administration alleviates rotavirus infection and immune response in the weaned piglet model. *Frontiers in Immunology*, *9*, 1654.

Marthaler, D., Homwong, N., Rossow, K., Culhane, M., Goyal, S., Collins, J., Matthijnssens, J. & Ciarlet, M. (2014a). Rapid detection and high occurrence of porcine rotavirus A, B, and C by RT-qPCR in diagnostic samples. *Journal of Virological Methods*, *209*, 30-34.

Marthaler, D., Rossow, K., Culhane, M., Collins, J., Goyal, S., Ciarlet, M., & Matthijnssens, J. (2013). Identification, phylogenetic analysis and classification of porcine group C rotavirus VP7 sequences from the United States and Canada. *Virology*, *446*(1-2), 189-198.

Marthaler, D., Rossow, K., Culhane, M., Goyal, S., Collins, J., Matthijnssens, J., Nelson, M., & Ciarlet, M. (2014b). Widespread rotavirus H in commercially raised pigs, United States. *Emerging Infectious Diseases*, *20*(7), 1203.

Matthijnssens, J., Attoui, H., Bányai, K., Brussaard, CP, Danthi, P., Del Vas, M., Dermody, T.S., Duncan, R., Fang, Q., Johne, R., & Wei, T. (2022). ICTV virus taxonomy profile: Sedoreoviridae 2022. *Journal of General Virology*, *103* (10), 001782.

Miyabe, F. M., Dall Agnol, A. M., Leme, R. A., Oliveira, T. E. S., Headley, S. A., Fernandes, T., Goncalves, A., Alfieri, A.F., & Alfieri, A. A. (2020). Porcine rotavirus B as primary causative agent of diarrhoea outbreaks in newborn piglets. *Scientific Reports*, *10*(1), 22002.

Monteagudo, L. V., Benito, A. A., Lázaro-Gaspar, S., Arnal, J. L., Martin-Jurado, D., Menjon, R., & Quílez, J. (2022). Occurrence of rotavirus A genotypes and other enteric pathogens in diarrheic suckling piglets from Spanish swine farms. *Animals*, *12*(3), 251.

Murao, L. A. E., Bacus, M. G., Junsay, N. X. T., Albarillo, D. L. D., Otero, M. C. B., Buenaventura, S. G. C., Ligue, K.D.B. & Alviola, P. A. (2019). Spatiotemporal dynamics and risk factors of rotavirus A circulation in backyard pig farms in a Philippine setting. *Tropical Animal Health and Production*, *51*, 929-937.

Nguyen, T. V., Yuan, L., Azevedo, M. S., Jeong, K. I., González, A. M., & Saif, L. J. (2007). Transfer of maternal cytokines to suckling piglets: in vivo and in vitro models with implications for immunomodulation of neonatal immunity. *Veterinary Immunology and Immunopathology*, *117*(3-4), 236-248.

Nyaga, M. M., Jere, K. C., Esona, M. D., Seheri, M. L., Stucker, K. M., Halpin, R. A., Akopov, A., & Mphahlele, M. J. (2015). Whole genome detection of rotavirus mixed infections in human, porcine and bovine samples co-infected with various rotavirus strains collected from sub-Saharan Africa. *Infection, Genetics and Evolution*, *31*, 321-334.

Papp, H., László, B., Jakab, F., Ganesh, B., De Grazia, S., Matthijnssens, J., Ciarlet, M., Martella, V & Bányai, K. (2013). Review of group A rotavirus strains reported in swine and cattle. *Veterinary microbiology*, *165*(3-4), 190-199.

Park, J.G., Alfajaro, M.M., Cho, E. H., Kim, J. Y., Soliman, M., Baek, Y.B., Lee, J. H., & Kang, M. I. (2019). Development of a live attenuated trivalent porcine rotavirus A vaccine against disease caused by recent strains most prevalent in South Korea. *Veterinary Research*, *50*, 1-16.

Patel, M. M., Pitzer, V. E., Alonso, W. J., Vera, D., Lopman, B., Tate, J., Vibound, C., & Parashar, U. D. (2013). Global seasonality of rotavirus disease. *The Pediatric Infectious Disease Journal*, *32*(4), e134-e147.

Qiao, M., Li, M., Li, Y., Wang, Z., Hu, Z., Qing, J., Huang, J., Jiang, J., Jiang, Y., Zhang, J., Gao, C., Yang, C., Li, X., & Zhou, B. (2024). Recent molecular characterization of porcine rotaviruses detected in China and their phylogenetic relationships with human Rotaviruses. *Viruses*, *16*(3), 453.

Saif, L., & Vlasova, A. (2022) Rotaviral diarrhoea in pigs. Pork Information Gateway. Retrieved from https://porkgateway.org/resource/rotaviral-diarrhea-in-pigs/

Santiana, M., Ghosh, S., Ho, B. A., Rajasekaran, V., Du, W. L., Mutsafi, Y., Jesus-Diaz, D. D., & Altan-Bonnet, N. (2018). Vesicle-cloaked virus clusters are optimal units for inter-organismal viral transmission. *Cell Host & Microbe*, *24*(2), 208-220.

Shepherd, F. K., Freeman, M. J., Culhane, M. R., Marthaler, D. G. (2019). Reoviruses (Rotaviruses and Reoviruses). In: Zimmerman, J. J., Karriker, L. A., Ramirez, A., Schwartz, K.J., Stevenson, G. W., & Zhang, J. (eds.). *Diseases of swine* (pp. 715-727), Wiley Online Library.

Shepherd, F. K., Herrera-Ibata, D. M., Porter, E., Homwong, N., Hesse, R., Bai, J., & Marthaler, D. G. (2018). Whole genome classification and phylogenetic analyses of rotavirus B strains

from the United States. *Pathogens*, *7*(2), 44.

Shepherd, F. K., Murtaugh, M. P., Chen, F., Culhane, M. R., & Marthaler, D. G. (2017). Longitudinal surveillance of porcine rotavirus B strains from the United States and Canada and in silico identification of antigenically important sites. *Pathogens*, *6*(4), 64.

Suzuki, T., & Inoue, D. (2018). Full genome-based genotyping system for rotavirus H and detection of potential gene recombination in nonstructural protein 3 between porcine rotavirus H and rotavirus C. *Journal of General Virology*, *99*(12), 1582-1589.

Theuns, S., Desmarets, L. M., Heylen, E., Zeller, M., Dedeurwaerder, A., Roukaerts, I. D., & Nauwynck, H. J. (2014). Porcine group A rotaviruses with heterogeneous VP7 and VP4 genotype combinations can be found together with enteric bacteria on Belgian swine farms. *Veterinary Microbiology*, *172*(1-2), 23-34.

Theuns, S., Vyt, P., Desmarets, L. M., Roukaerts, I. D., Heylen, E., Zeller, M., Matthijnssens, J. & Nauwynck, H. J. (2016). Presence and characterization of pig group A and C rotaviruses in faeces of Belgian diarrheic suckling piglets. *Virus Research*, *213*, 172-183.

Tian, G., Liang, X., Chen, D., Mao, X., Yu, J., Zheng, P., Huang, Z., & Yu, B. (2016). Vitamin D3 supplementation alleviates rotavirus infection in pigs and IPEC-J2 cells via regulating the autophagy signalling pathway. *The Journal of Steroid Biochemistry and Molecular Biology*, *163*, 157-163.

Tuanthap, S., Phupolphan, C., Luengyosluechakul, S., Duang-In, A., Theamboonlers, A., Wattanaphansak, S., Vongpunsawad, S., Amonsin, A. & Poovorawan, Y. (2018). Porcine rotavirus C in pigs with gastroenteritis on Thai swine farms, 2011–2016. *PeerJ Microbiology*, *6*, e4724.

VinodhKumar, O. R., Sircar, S., Pruthvishree, B. S., Nirupama, K. R., Singh, B. R., Sinha, D. K., Rupner, R., & Malik, Y. S. (2020). Cross-sectional study on rotavirus A (RVA) infection and assessment of risk factors in pre-and post-weaning piglets in India. *Tropical Animal Health and Production*, *52*, 445-452.

Vlasova, A. N., Amimo, J. O., & Saif, L. J. (2017). Porcine rotaviruses: epidemiology, immune responses and control strategies. *Viruses*, *9*(3), 48.

Wu, F. T., Bányai, K., Jiang, B., Liu, L. T. C., Marton, S., Huang, Y. C., Huang, L.M., Liao, M.H. & Hsiung, C. A. (2017). Novel G9 rotavirus strains co-circulate in children and pigs, Taiwan. *Scientific Reports*, *7*(1), 40731.

Wu, F. T., Liu, L. T. C., Jiang, B., Kuo, T. Y., Wu, C. Y., & Liao, M. H. (2022). Prevalence and diversity of rotavirus A in pigs: Evidence for a possible reservoir in human infection. *Infection, Genetics and Evolution*, *98*, 105198.